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Quinclorac Absorption and Translocation Characteristics in Quinclorac- and Propanil-Resistant and -Susceptible Barnyardgrass (*Echinochloa crus-galli*) Biotypes

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Studies were initiated to evaluate absorption, translocation, and distribution of ^{14}C-quinclorac in propanil- and quinclorac-resistant (R-BYG) and -susceptible (S-BYG) barnyardgrass. No differential absorption of ^{14}C-quinclorac was observed between R-BYG and S-BYG, but more ^{14}C remained in the treated leaf of S-BYG (57% of total absorbed) compared with the R-BYG leaf (34% of total absorbed) at 72 h after treatment (HAT). After 12 HAT, 20 and 15% of the amount absorbed had been translocated basipetally by R-BYG and S-BYG, respectively. At 72 HAT, 27 and 17% of the total absorbed ^{14}C had been translocated acropetally by R-BYG and S-BYG, respectively. The levels of ^{14}C above the treated leaf continued to increase throughout the duration of the experiment in R-BYG while levels of ^{14}C above the treated leaf in S-BYG remained relatively constant. Seven percent more of the total absorbed ^{14}C was exuded from roots of R-BYG than S-BYG at 72 HAT. Although differential translocation was observed between R-BYG and S-BYG, it is unclear whether this difference is a cause of quinclorac resistance or an effect of some other physiological process. Further research is needed to determine if differential translocation is due to metabolism or other physiological factors.
Nomenclature: Quinclorac; barnyardgrass, *Echinochloa crus-galli* (L.) Beauv. ECHCG.
Key words: Auxin herbicide, auxinic herbicide, differential translocation, herbicide resistance.

Recently, a barnyardgrass biotype from Arkansas was confirmed to have multiple resistance to the herbicides quinclorac and propanil (Lovelace et al. 2000). Currently, the mechanisms of herbicide resistance in this biotype have not been reported. Generally, herbicide resistance in plants can be attributed to reduced uptake, increased translocation, or metabolic degradation of these agrochemicals. For example, the mechanism of resistance for eastern black nightshade (*Solanum ptycanthum* Dun.) to nicosulfuron was attributed to reduced translocation (Carey et al. 1997a). Differential absorption and translocation is thought to be a major factor influencing glufosinate resistance in several weed species (Steckel et al. 1997). Differences in translocation and metabolism have also been shown to be important in the susceptibility of broadleaf signalgrass [*Urochloa platyphylla* (Nash) R.D. Webster] to primisulfuron and nicosulfuron (Gallaher et al. 1999).

Rapid foliar uptake of ^{14}C-quinclorac was observed in southern crabgrass [*Digitaria ciliaris* (Retz.) Koel.] and Kentucky bluegrass (*Poa pratensis* L.) (Chism et al. 1991). These species are susceptible and resistant to quinclorac, respectively. Within 0.5 h, 85 and 66% of the applied quinclorac was absorbed by leaves of southern crabgrass and Kentucky bluegrass, respectively. Over time, more quinclorac was translocated into nontreated Kentucky bluegrass leaves than into nontreated southern crabgrass leaves. Chism et al. (1991) suggested that reduced transport of quinclorac in susceptible species may be due to the rapid phytotoxic activity in these susceptible plants. By 128 h after treatment (HAT), 17% of the applied quinclorac was exuded by roots of Kentucky bluegrass into a nutrient solution, whereas little root exudation occurred in southern crabgrass. No differences in quinclorac metabolism were detected in these two species; thus it was concluded that distribution, dilution, and exudation were the probable mechanisms for quinclorac selectivity.

Absorption of sublethal rates of quinclorac into leafy spurge (*Euphorbia esula* L.), a quinclorac-susceptible dicotyledonous species, was 79% at 2 d after treatment (DAT) (Lamoureux and Rusness 1995). The treated leaf contained 26% of the applied quinclorac, while 11% was exuded by roots into soil. By 21 DAT, only 44% remained in the plant and 49% had been transported to the root and exuded. It was also suggested that quinclorac remaining in the leaves was sequestered, thus reducing phytotoxicity (Lamoureux and Rusness 1995). Generally, reports indicate that some quinclorac exudation can occur from roots of both resistant and susceptible plants.

Although differential absorption and translocation can influence herbicide resistance in some species, these parameters have not been shown to be factors that influence resistance to quinclorac in barnyardgrass biotypes from Mississippi (Grossmann and Kwiatkowski 2000) or Spain (Lopez-Martinez and De Prado 1996). These studies indicated that differential acropetal and basipetal translocation of ^{14}C-quinclorac was not significantly different between the quinclorac-resistant and -susceptible barnyardgrass biotypes. Most of the quinclorac remained in the treated leaf, and exudation of quinclorac into the nutrient solution was negligible (Lopez-Martinez and De Prado 1996).

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Absorption and translocation of quinclorac in resistant and susceptible biotypes of false cleavers (*Galium spurium* L.) was also not different (Van Eerd et al. 2005). The majority of the quinclorac absorbed by false cleavers was translocated out of the treated leaf, with varying amounts found above the treated leaf, below the treated leaf, and in the roots. Some quinclorac was detected in the root exudates of false cleavers (Van Eerd et al. 2005).

In addition to absorption and translocation, metabolism also influences herbicide resistance in plants. Reports have shown that metabolism of quinclorac in barnyardgrass and other monocot species was about 10% after being exposed to ^{14}C-quinclorac for 7 h (Grossmann and Kwiatkowski 2000). Quinclorac was metabolized into two unidentified compounds in these grass species. No significant differences were found in the amount of quinclorac metabolism between resistant and susceptible biotypes of barnyardgrass and *Echinochloa hispidula*. In Kentucky bluegrass and southern crabgrass, metabolism of quinclorac into a single unidentified soluble compound was very low and did not differ in these two species (Chism et al. 1991). Similarly, quinclorac metabolism was not detected in large crabgrass [*Digitaria sanguinalis* (L.) Scop.], a susceptible species, nor in goosegrass [*Eleusine indica* (L.) Gaertn.], a resistant species (Zawierucha and Penner 2000). Although some metabolism of quinclorac may occur in monocots, metabolism is generally low and does not appear to influence resistance. In addition, most of the quinclorac absorbed into monocots appears to remain in the parent form.

Abdalla et al. (2006) concluded that smooth crabgrass [*Digitaria ischaemum* (Schreb.) Schreb. ex Muhl.] resistance to quinclorac was due to a disruption in the pathway leading to the induction of 1-aminocyclopropane-1-carboxylic acid synthase and ethylene biosynthesis. Goss and Dyer (2003) reported that kochia [*Kochia scoparia* (L.) Schrad.] resistance to dicamba was due to a mutation in an auxin binding protein.

Although some mechanisms of auxin herbicide resistance have been proposed, no evidence has been provided to indicate the mechanism of quinclorac resistance of this new multiple-herbicide-resistant barnyardgrass biotype from Arkansas.

The objective of this research was to determine if differential absorption or translocation of quinclorac could be linked to resistance in the multiple-herbicide-resistant (propanil and quinclorac) barnyardgrass biotype recently discovered in Arkansas (Lovelace et al. 2000).

Materials and Methods

Seeds from a barnyardgrass population suspected of quinclorac and propanil resistance were collected from a rice field in Craighead County, Arkansas in 1999. An initial screening test was initiated October 19, 1999 on barnyardgrass plants at the four-leaf growth stage. Plants were treated with standard registered rates of quinclorac (420 g ai/ha) plus nonionic surfactant[1] (0.25% v/v), propanil (4.48 kg ai/ha), or a combination of quinclorac plus propanil (no surfactant) at these respective rates. Plants exhibited little response from application of these herbicides applied alone or in combination (data not shown). Because of the limited quantity of seeds, barnyardgrass plants treated with quinclorac plus propanil were grown to maturity for seed increase. These seeds were the source of quinclorac- and propanil-resistant barnyardgrass plants used in our tests. A susceptible barnyardgrass population is maintained at the University of Arkansas and periodically tested to ensure susceptibility. These seeds were the source of quinclorac- and propanil-susceptible barnyardgrass plants used in our tests.

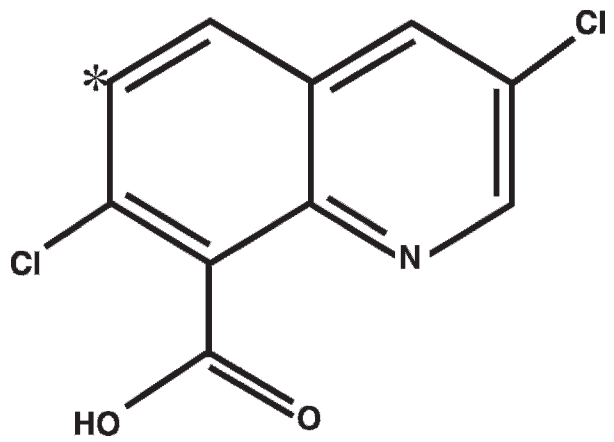

Figure 1. Structure of quinclorac. * Denotes location of radiolabeled carbon.

Tests were initiated on September 1 and November 4, 2001 to evaluate differential absorption and translocation of quinclorac in propanil- and quinclorac-resistant barnyardgrass (R-BYG) and susceptible barnyardgrass (S-BYG) biotypes. Tests were conducted twice, with each test conducted as a randomized complete block with four replications, and treatments were arranged as a two-factor factorial (biotype by time of harvest). R-BYG and S-BYG seeds (from the populations described previously) were germinated in vermiculite, and seedlings were transferred to a hydroponic growth media in 350-ml cups containing 175 ml of full-strength Hoagland's solution (Hoagland and Arnon 1938). Hoagland's solution was added daily as needed and pH and conductivity were measured to ensure that salts were not accumulating. Quinclorac treatments were applied to plants at the four-leaf growth stage.

Plants were treated with a broadcast application of quinclorac (Facet 75 DF®) at 0.21 kg ai/ha and nonionic surfactant at 0.25% v/v using a track sprayer in an enclosed spray chamber calibrated to deliver 187 L/ha of spray solution. All plants were then transported to an isotope laboratory where ^{14}C-quinclorac[2] (ring B, no. 6-^{14}C) (specific activity 83.47 MBq/mg; Figure 1) was applied. Four-leaf plants were treated with four 1-µl droplets of ^{14}C-quinclorac formulated in combination with the spray solution (Facet 75 DF and surfactant) to give 416.67 Bq/µl, and a final herbicide concentration of 13.3 mM. The adaxial surface of the third leaf was treated with four 1-µl droplets of the spotting solution using a digital microsyringe.[3] After treatment, plants were placed in a growth chamber (30/20 C; 16-h days; photosynthetic photon flux density of 500 µmol m^{-2} s^{-1}; 95% ± 5% relative humidity). At 0, 3, 6, 12, 24, 48, and 72 HAT, plants

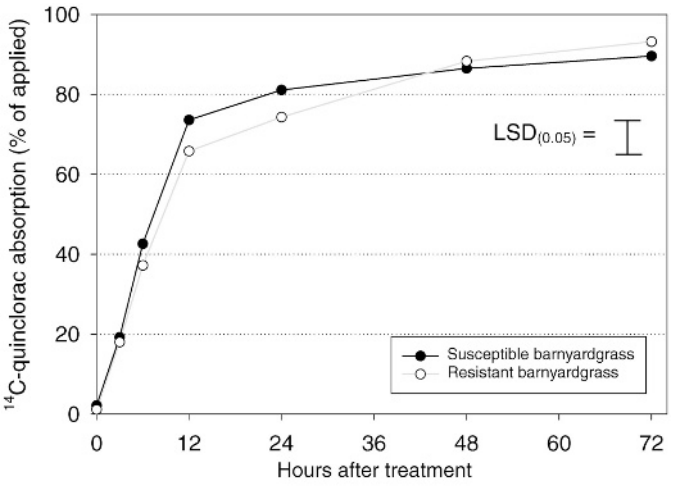

Figure 2. Absorption of ^{14}C-quinclorac by propanil- and quinclorac-resistant (R-BYG) and -susceptible barnyardgrass (S-BYG) plants over time. No differences were detected between biotypes at any time. $LSD_{(0.05)}$ bar to make comparisons between biotypes at a particular time interval.

were divided into treated leaf, leaves above the treated leaf, leaves below the treated leaf, and roots.

At harvest, the treated leaf was rinsed with 4 ml of deionized water in a 20-ml scintillation vial for 15 s to remove unabsorbed ^{14}C-quinclorac. Each leaf wash vial was then supplied with 15 ml of scintillation cocktail[4] and analyzed for ^{14}C-quinclorac by liquid scintillation spectrometry (LSS).[5] Four milliliters of the hydroponic growing medium was taken from each plant and placed in a 20-ml scintillation vial with 15 ml of scintillation cocktail and analyzed for ^{14}C to determine possible exudation of ^{14}C (parent herbicide or possible ^{14}C metabolites) from the roots. The results of the 4-ml extracts were extrapolated to represent the total volume of solution in the cups (175 ml). All plant tissue was oxidized using a biological oxidizer,[6] and $^{14}CO_2$ was trapped in a CO_2 trapping solution.[7] The solution was evaluated for $^{14}CO_2$ using LSS. Recovery of ^{14}C was about 94% (data not shown).

Data Analysis. Data were subjected to analysis of variance with partitioning appropriate for a factorial arrangement of treatments, and means were separated using Fisher's Protected LSD at the 5% level of probability. There was no interaction of factors among runs; therefore, data for both studies were pooled over runs. All analyses were conducted using PROC MIXED.[8]

Results and Discussion

Absorption of ^{14}C-quinclorac into the treated leaf was similar for the R-BYG and S-BYG biotypes (Figure 2). No differences in absorption among barnyardgrass biotypes were detected at any observation time, which has also been reported by Lopez-Martinez and De Prado (1996) and by Grossmann and Kwiatkowski (2000) using other barnyardgrass biotypes. For both of our biotypes, ^{14}C-quinclorac absorption was very rapid up to 12 HAT, with 66 and 73% of the applied ^{14}C-quinclorac being absorbed by R-BYG and S-BYG, respectively (Figure 2). By 72 HAT, 93 and 90% of the total applied ^{14}C-quinclorac had been absorbed by R-BYG and S-BYG, respectively.

Although the absorption of ^{14}C-quinclorac did not differ between the two biotypes, distribution of the ^{14}C-quinclorac (or possible ^{14}C metabolites) within the two biotypes was different (Table 1). ^{14}C levels in the treated leaf were similar at 3 HAT with both biotypes, but differences in ^{14}C retention within the treated leaf became evident at 6 HAT. At this sampling time, 73 and 62% of the total absorbed ^{14}C remained in the treated leaf of the S-BYG and R-BYG,

Table 1. Translocation of radioactivity in propanil- and quinclorac-resistant and -susceptible barnyardgrass biotypes treated with foliar-applied ^{14}C-quinclorac.

HAT[a]	Barnyardgrass biotype	Distribution of radioactivity				
		Treated leaf	Above treated leaf	Below treated leaf	Roots	Exudate[b]
		% of absorbed				
3	Resistant	80	5	10	2	3
	Susceptible	82	4	10	2	2
6	Resistant	62	18	13	3	4
	Susceptible	73	8	13	2	4
12	Resistant	57	18	20	1	4
	Susceptible	66	15	15	1	3
24	Resistant	54	20	17	2	7
	Susceptible	64	14	15	2	5
48	Resistant	45	24	16	2	13
	Susceptible	59	18	14	1	8
72	Resistant	34	27	20	2	17
	Susceptible	57	17	14	2	10
	$LSD_{(0.05)}$[c]	6	5	4	NS	3

[a] Abbreviation: HAT, h after treatment; NS, not significant.

[b] Exudate indicates percentage of total absorbed radioactivity detected in nutrient solution.

[c] $LSD_{(0.05)}$ values are to make comparisons of percentage absorbed radioactivity between biotypes at a single observation time (HAT) or to make comparisons of percentage absorbed radioactivity between different observation times for a single biotype. All $LSD_{(0.05)}$ values are to make comparisons within their specified column.

respectively. ^{14}C continued to be translocated out of the treated leaf of R-BYG faster than in S-BYG, which is indicated by 1.7-fold less remaining in the treated leaf of R-BYG at 72 HAT. Differences in translocation between R-BYG and S-BYG may be due to quinclorac sequestration of the herbicide *in planta* or affinity of quinclorac for binding sites within the treated leaf.

The difference in translocation of ^{14}C-quinclorac between our two biotypes is inconsistent with previous findings, which showed no differential translocation between other quinclorac-resistant and -susceptible barnyardgrass biotypes (Grossmann and Kwiatkowski 2000; Lopez-Martinez and De Prado 1996). In our experiments, plants were treated with a broadcast application of nonradiolabeled quinclorac at 0.21 kg/ha in combination with ^{14}C-quinclorac (13 mM final concentration), whereas in the cited experiments the total quinclorac concentration was sublethal (100 μM final concentration) (Grossmann and Kwiatkowski 2000; Lopez-Martinez and De Prado 1996). In our experiments, quinclorac treatment at 0.21 kg/ha may have caused rapid localized toxicity that reduced quinclorac translocation in S-BYG. Toxic levels of herbicides can influence their translocation in plant tissues (Chism et al. 1991; Devine 1989). In addition, quinclorac is an auxin-type herbicide, and some herbicides with this mode of action can limit transport through induced swelling of plant tissues in susceptible species, causing disruption of phloem, xylem, and cambium (Meyer 1970). Thus the quinclorac application rate may affect the movement and distribution of ^{14}C-quinclorac within the plant.

Leaves above the treated leaf were the largest reservoirs for ^{14}C translocation out of the treated leaf for both the S-BYG and R-BYG biotypes (Table 1). More ^{14}C moved into leaves above the treated leaf in R-BYG than in S-BYG. The levels of ^{14}C above the treated leaf of R-BYG continued to increase to 27% through 72 HAT. Maximum ^{14}C levels above the treated leaf of the S-BYG plants were 18% of the total absorbed radioactivity, and occurred 48 HAT. Other research has shown that high levels of quinclorac and its metabolites (glucose and pentosylglucose esters of quinclorac) accumulated in the apex of leafy spurge, also indicating that the apex and new leaves were reservoirs for quinclorac and its metabolites (Lamoureux and Rusness 1995).

Leaf tissue below the treated leaf was also a reservoir for ^{14}C moving out of the treated leaf. Leaves below the treated leaf contained 20 and 15% of the total absorbed ^{14}C at 12 HAT in R-BYG and S-BYG, respectively (Table 1). Although a significant amount of ^{14}C moved below the treated leaf by 12 HAT, levels remained relatively constant in both biotypes throughout the remainder of the test. In the R-BYG, the greater quantities of ^{14}C below the treated leaf may have been translocated into this region via assimilate flow as compared with the S-BYG, where quinclorac may have been immobilized in the treated leaf.

Negligible accumulation of ^{14}C was detected in the roots of either barnyardgrass biotype after foliar application of ^{14}C-quinclorac. However, the roots served as a pathway for quinclorac exudation into the hydroponic growth media. Exudation of ^{14}C continued from both biotypes through time, but was greater in R-BYG (17% exudation of the total absorbed ^{14}C) than in S-BYG (10% of total) at 72 HAT. Our results were similar to those reported by Chism et al. (1991), who indicated that 17% of the applied quinclorac was exuded by roots of Kentucky bluegrass (a quinclorac-resistant species) into a hydroponic solution by 128 HAT, whereas little root exudation occurred in southern crabgrass (a quinclorac-susceptible species). Furthermore, reports indicated that quinclorac was rapidly exuded from leafy spurge roots (a quinclorac-susceptible species) (Lamoureux and Rusness 1995), and 2,4-D was exuded from roots of jimsonweed (susceptible to 2,4-D) (Fites et al. 1964). In contrast, our results did not reflect findings of a previous report indicating that no significant exudation from quinclorac-resistant and -susceptible barnyardgrass occurred (Lopez-Martinez and De Prado 1996). It is unknown whether exudation from the R-BYG and S-BYG occurred because of diffusion out of the symplast, or to herbicide damage to the tissue (Devine 1989).

Several examples have been presented here illustrating the occurrence of differential translocation of herbicides by a variety of plant species. The more rapid movement of ^{14}C (within the plant and via exudation) in our R-BYG could be due to an inherent increase in translocation or to greater mobility of quinclorac metabolites. Our studies reported here did not examine metabolism as a possible resistance mechanism in this quinclorac-resistant barnyardgrass biotype. However, since quinclorac has been shown to be metabolized very slowly in other resistant monocots (Chism et al. 1991), and this compound is not degraded to a significant degree by photolysis (Vencill 2002), degradation products and increased metabolism of ^{14}C-quinclorac is an unlikely explanation. Thus, the major portion of ^{14}C found in the nutrient solution and in various parts of the plant is most likely ^{14}C-quinclorac. On the other hand, increased metabolism of propanil has been shown to be the resistance mechanism in another barnyardgrass biotype from Arkansas found to be resistant to high rates of propanil (Carey et al. 1997b; Norsworthy et al. 1998).

Overall, the explanation put forward on kochia resistance to dicamba may be most pertinent to our findings with our propanil- and quinclorac-resistant barnyardgrass biotype. Kochia resistance to dicamba is thought to arise from an alteration in the auxin binding protein (Goss and Dyer 2003). The auxin binding proteins within kochia have a lower affinity for dicamba and endogenous auxins, rendering these plants resistant. If a mutation in the auxin binding protein in our R-BYG has occurred, quinclorac may not be interacting with the altered auxin binding proteins within the treated leaf, thus allowing the herbicide to move more freely throughout the plant via the assimilate stream. Conversely, quinclorac may be readily interacting with the unaltered auxin binding proteins within S-BYG, and thus more quinclorac is bound into herbicide–protein complexes, restricting herbicide movement within the plant. Additional research will be required to elucidate the actual quinclorac resistance mechanism in this propanil- and quinclorac-resistant barnyardgrass biotype.

The recent development and expansion of quinclorac- and propanil-resistant barnyardgrass in Arkansas seriously compromises the utility of two of the most common herbicides used in rice for control of this species. The presence of multiple-herbicide-resistant barnyardgrass biotypes in rice

fields complicates weed management issues and potentially increases rice production costs. Understanding the mechanisms of resistance will provide researchers and producers with a better understanding of alternative weed management strategies.

Sources of Materials

[1] Latron AG-98 nonionic surfactant. Dow Agrosciences LLC, 9330 Zionsville Rd., Indianapolis, IN 46268.

[2] ^{14}C-Quinclorac. BASF Corporation, Agricultural Products Group, P.O. Box 13528, Research Triangle Park, NC 27709.

[3] 1700 Series Gastight Digital Syringe. Hamilton Company, 4970 Energy Way, Reno, NV 89502.

[4] Ultima Gold XR High Flashpoint Scintillation Cocktail. Packard Instrument Co., 800 Research Parkway, Meriden, CT 06450.

[5] Packard TriCarb 2900 TR Liquid Scintillation Analyzer. Packard Instrument Co., 800 Research Parkway, Meriden, CT 06450.

[6] OX-700 Biological Oxidizer. R. J. Harvey Instrument Corporation, 123 Patterson St., Hillsdale, NJ 07642.

[7] Harvey Carbon-14 Cocktail. R. J. Harvey Instrument Corporation, 123 Patterson St., Hillsdale, NJ 07642.

[8] SAS®, version 8, SAS Institute, 100 Campus Drive, Cary, NC 27513.

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Literature Cited

Abdallah, I., A. J. Fischer, C. L. Elmore, M. E. Saltveit, and M. Zaki. 2006. Mechanism of resistance to quinclorac in smooth crabgrass (*Digitaria ischaemum*). Pestic. Biochem. Physiol. 84:38–48.

Carey, J. B., D. Penner, and J. J. Kells. 1997a. Physiological basis for nicosulfuron and primisulfuron selectivity in five plant species. Weed Sci. 45:22–30.

Carey, V. F., R. E. Hoagland, and R. E. Talbert. 1997b. Resistance mechanism of propanil-resistant barnyardgrass: II. *In-vivo* metabolism of the propanil molecule. J. Pestic. Sci. 49:333–338.

Chism, W. J., S. W. Bingham, and R. L. Shaver. 1991. Uptake, translocation, and metabolism of quinclorac in two grass species. Weed Technol. 5:771–775.

Devine, M. D. 1989. Phloem translocation of herbicides. Rev. Weed Sci. 4: 191–213.

Fites, R. C., F. W. Slife, and J. B. Hanson. 1964. Translocation and metabolism of radioactive 2,4-D in jimsonweed. Weeds 12:180–183.

Gallaher, K., T. C. Mueller, R. M. Hayes, O. Schwartz, and M. Barrett. 1999. Adsoprtion, translocation, and metabolism of primisulfuron and nicosulfuron in broadleaf signalgrass (*Brachiaria platyphylla*) and corn. Weed Sci. 47:8–12.

Goss, G. A. and W. E. Dyer. 2003. Physiological characterization of auxinic herbicide-resistant biotypes of kochia (*Kochia scoparia*). Weed Sci. 51:839–844.

Grossmann, K. and J. Kwiatkowski. 2000. The mechanism of quinclorac selectivity in grasses. Pestic. Biochem. Physiol. 66:83–91.

Hoagland, D. R. and D. I. Arnon. 1938. The water-culture method for growing plants without soil. Circ. 347, California Agricultural Experiment Station.

Lamoureux, G. L. and D. G. Rusness. 1995. Quinclorac absorption, translocation, metabolism, and toxicity in leafy spurge (*Euphorbia esula*). Pestic. Biochem. Physiol. 53:210–226.

Lopez-Martinez, N. and R. De Prado. 1996. Fate of quinclorac in resistant *Echinochloa crus-galli*. Pages 535–540 *in* Second International Weed Control Congress. Copenhagen, Denmark.

Lovelace, M. L., J. D. Reaper, E. F. Scherder, L. A. Schmidt, and R. E. Talbert. 2000. Multiple resistance of propanil-resistant barnyardgrass (*Echinochloa crus-galli*) to quinclorac. Abstr. Proc. 28th Rice Tech. Work. Group. 28:153.

Meyer, R. E. 1970. Picloram and 2,4,5-T influence on honey mesquite morphology. Weed Sci. 18:525–231.

Norsworthy, J. K., Talbert, R. E., and Hoagland, R. E.. 1998. Chlorophyll fluorescence for rapid detection and confirmation of propanil-resistant barnyardgrass (*Echinochloa crus-galli*). Weed Sci. 46:163–169.

Steckel, G. J., S. E. Hart, and L. M. Wax. 1997. Absorption and translocation of glufosinate on four weed species. Weed Sci. 45:378–381.

Van Eerd, L. L., G. R. Stephenson, J. Kwiatkowski, K. Grossmann, and C. Hall. 2005. Physiological and biochemical characterization of quinclorac resistance in a false cleavers (*Galium spurium* L.) biotype. J. Agric. Food Chem. 53:1144–1151.

Vencill, W. K. 2002. Quinclorac. Pages 386–387 *in* W. K. Vencill, ed. Herbicide Handbook. 8th ed. Lawrence, KS: Weed Science Society of America.

Zawierucha, J. A. and D. Penner. 2000. Absorption, translocation, metabolism, and spray retention of quinclorac in *Digitaria sanguinalis* and *Eleusine indica*. Weed Sci. 48:296–301.

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